

EDITORIAL

Mental disorder and the strategies of prevention^{1, 2}

Prevention and health promotion are in the news. General practitioners in the UK must offer health checks or risk a financial penalty; 1993 will see the second World Congress of Preventive Cardiology; the Health Education Authority battles against smoking and AIDS; and so on in many branches of medicine – but not in psychiatry. ‘Preventive psychiatry’ has not yet arrived. This is surprising, for psychiatry is confronted by many incurable patients and it was among the first specialities to embrace epidemiological research.

Throughout medicine the understanding of prevention has been hindered by the tacit belief that disease and normality are separate entities. In psychiatry, whether the discussion is of Alzheimer’s ‘disease’, depression or schizophrenia, the same question is asked, ‘Has this patient got it?’, the answer being ‘yes’ or ‘no’. Researchers, presented with a distribution of scores for the General Health Questionnaire (GHQ) or a depression inventory, hurry to apply an arbitrary cut-off point in order to separate those whom clinicians will accept as ‘cases’ from the remainder, who are grouped together as ‘normals’. As Pickering (1968) wrote, ‘Medicine in its present state can count up to two but not beyond’.

No such separation really exists. As the old man remarked to his wife, ‘All the world is queer save thee and me, and even thou art a little queer’. In those few reports which do present complete distributions of scores for the GHQ, depression or dementia (Gurland *et al.* 1983; Brenner, 1985; Brayne & Calloway, 1988; Anderson *et al.* 1993) then – just as with IQ scores – they form a continuum. Case definitions are necessary wherever management decisions are dichotomous (‘treat’ or ‘not treat’); but they should be recognized for what they are, which is an operational convenience and not a description of nature. Disease comes in all sizes, and we should move away from asking ‘Has this person got it?’ towards ‘How much of it do they have?’ Recognition of the continuous distribution which unites the whole population, sick and healthy, is a first and necessary step towards rational prevention.

The continuum between disease and normality is not readily apparent in hospitals and clinics, whose patients are the survivors of a selection process whereby only the more severely affected will be referred, thus creating an illusion of a qualitative separation of disease from normality. Patients in general practice have not been subjected to this process, and ‘a touch of depression’ is seen to be all too common.

Awareness of the ‘iceberg phenomenon’ has stimulated enthusiasm for screening. This, however, is simply the pursuit of earlier diagnosis and it should not be confused with prevention. Clinicians find it natural to turn to the ‘high-risk strategy’ (Rose, 1985), which offers special support to the most susceptible or exposed individuals. This approach has been applied vigorously in cardiovascular disease through the identification and control of its various risk factors, and it has come to dominate the whole medical approach to prevention. Its purpose is to truncate the risk factor distribution by identifying and eliminating hypertension, drug abuse, criminal violence and so on, while leaving the rest of the population in peace. Such a truncation has never yet been achieved. The strategy could be compared with an attempt to control icebergs by sending warships to shoot off their visible portions, or with famine relief which feeds the hungry but does not tackle the causes of famine. The high-risk strategy provides succour for some needy individuals, but its

¹ The general issues discussed in this editorial are dealt with more fully in *The Strategy of Preventive Medicine* by G. Rose. Oxford University Press: Oxford, 1992.

² Address for correspondence: Professor G. Rose, Trevalley, Penfold Lane, Holmer Green, High Wycombe, HP15 6XS.

scope is limited by the underlying false assumptions that the problem is confined to a distinct minority and that it can be considered in isolation from the parent population.

The weakness of the high-risk strategy reflects our poor ability to predict which individuals will become sick. Risk factors may identify a group with a much increased relative risk, but most high-risk individuals are likely to remain well and most clinical cases occur in those who were not at conspicuous risk. It is a matter of simple arithmetic: 'a large number of people exposed to a small risk commonly generate many more cases than a small number exposed to a high risk' (Rose, 1992). A broader, population-based approach to prevention is logical, realistic and potentially far more effective.

Not only do most clinical cases occur in low-risk individuals but also sub-clinical degrees of abnormality generate much morbidity. Using data from an American survey by Brenner (1985) it appears (Rose, 1992) that as much as three-quarters of depression-related social disability may arise in those whose scores on a depression inventory fall below the accepted threshold for a case. A 'touch of depression' may be only a small burden on the affected individuals but it is bad news for the community. To be effective, prevention must address the whole range of the problem.

The occurrence of disease, whether conspicuous or sub-clinical, cannot be understood except in the context of a distributional shift of risk factors and disease status involving the whole population (Rose & Day, 1990). The US/UK Cross-National Geriatric Study (Gurland *et al.* 1983) found that the prevalence of 'dementia' was about one-third higher in New York than London, the reason being a community-wide shift in the whole distribution of cognitive performance. 'Why was there more dementia in the New York men?' is the wrong question. The right question is, 'Why was the whole range of performance worse in this community?'. The prevalence of clinical dementia is just one aspect of influences which bear on the whole community, and prevention of frank as well as subclinical disorder depends on identifying and controlling those influences.

Aetiology needs to move beyond studying the characteristics of cases to finding the characteristics of sick populations. In a world-wide survey it was found (Rose & Day, 1990) that the prevalence of heavy drinking in a population could be predicted precisely from its average alcohol intake ($r = 0.97$). Similarly, the large regional variations in average GHQ scores within the UK can be used to predict with some accuracy the differences in prevalence of 'cases' ($r = 0.92$).

Several important conclusions follow. First, there is a characteristic of a community considered as a whole, namely, its overall mental health; this can be estimated by its average GHQ score and it is socially important in its own right. Secondly, the large inter-population differences in this attribute result from shifts of the entire distribution, to which the differences in case prevalence seem to be secondary. The visible part of the iceberg (prevalence) is a function of its total mass (the population average), and the one cannot be reduced without the other.

More data are needed in order to discover the extent of this phenomenon. It has been established in regard to alcohol intake, senile dementia, and GHQ scores (as well as in fields outside psychiatry); it may well apply also to aggression and depression.

The implications for prevention are clear. Hitherto control efforts have been focused almost exclusively on the deviant minorities (depressives, alcoholics and other drug abusers, those with behavioural disorders, the criminally aggressive, and so on). This alienation of the extremists is attractive to public and politicians, since it affirms the normality and innocence of the majority; but as a basis for prevention it has been a failure. Attention should now move towards understanding and perhaps controlling the mass determinants of population means, prevalence rates and incidence rates.

What is the psychiatric counterpart of the identification and control of water pollution, which so impressively reduced the incidence of cholera? To answer this question calls for research that bridges clinical skills, epidemiology and social studies; and consequent preventive action will likewise bridge medicine, social policy and politics. 'Preventive psychiatry' could have an exciting future.

G. ROSE

REFERENCES

- Anderson, J., Huppert, F. & Rose, G. (1993). Normality, deviance and minor psychiatric morbidity in the community. *Psychological Medicine* **23**, 475–485.
- Brayne, C. & Calloway, P. (1988). Normal ageing, impaired cognitive function and senile dementia of the Alzheimer's type: a continuum? *Lancet* *i*, 1265–1267.
- Brenner, B. (1985). Continuity between the presence and absence of the depressive syndrome. Paper presented at the 113th Annual Meeting of the American Public Health Association, Washington, DC, November, 1985. (Cited in Rose, G. *The Strategy of Preventive Medicine*, pp. 25–27. Oxford University Press: Oxford, 1992.)
- Gurland, B., Copeland, J., Kuriansky, J., Kelleher, M., Sharpe, L. & Dean, L. L. (1983). *The Mind and Mood of Aging. Mental Health Problems of the Community Elderly in New York and London*. Haworth Press: New York.
- Pickering, G. W. (1968). *High Blood Pressure*, 2nd edn. Churchill: London.
- Rose, G. (1985). Sick individuals and sick populations. *International Journal of Epidemiology* **14**, 32–38.
- Rose, G. (1992). *The Strategy of Preventive Medicine*. Oxford University Press: Oxford.
- Rose, G. & Day, S. (1990). The population mean predicts the number of deviant individuals. *British Medical Journal* **301**, 1031–1034.